angiography demonstrated that, at the site of successful radiofrequency ablation (RFA) of the AP, the catheter tip was outside the AV ring and beyond the angiographic atrial boundaries (manual contrast injection does not fill atrial appendages). The AP related to a PIARAA ended at the epicardial LV free-wall since there was a 50 ms prolongation of the V-A during reciprocating tachycardia with left bundle branch block aberrancy. At the successful RFA site an AP potential (K) was recorded in all cases. The RFA of the LAA-related AP was performed using a transeptal approach. One Pt had undergone 3 unsuccessful RFA attempts at two other centres.

	#App	Wmed	Tmed/max	K-delta
RAA-RV (1)	1	23	64/68	10
RAA-RV (2)	2	8	68/72	25
PIARA-LV	2	16	64/68	30
LAA-LV	6	47	47/66	NA

* Kent-Local auriculogram: 15 ms

Conclusions: RFA of AP's connecting atrial appendages and the epicardial ventricular surface, is feasible requiring not very many current applications (#App). Manual angiography enables us to demonstrate that the tip of the ablation catheter was located "outside the heart", in the respective appendages.

1123-163 Differences in Location and Electrophysiologic Properties of Atrial Tachycardias in Structurally Normal and Abnormal Hearts

R.J. Verdino, M.C. Burke, D.E. Kopp, J.G. Kall, P.A. Cooke, C.T. Johnson, A.C. Lin, D.J. Wilber. *University of Chicago, Chicago, IL, USA*

To determine if the characteristics of atrial tachycardias (AT) differ in patients with and without structural heart disease (SHD), we analyzed data from 47 consecutive patients undergoing successful ablation of 58 distinct ATs. Of the 20 pts with SHD, 2 had congenital heart disease, 4 coronary artery disease, 5 valvular heart disease, 5 hypertensive heart disease, and 4 non-ischemic cardiomyopathy. Nine pts had depressed left ventricular ejection fractions. Of the 58 ATs, 25 (43%) were located on the crista terminalis, 18 (31%) on the tricuspid annulus, 7 (12%) on the right atrial free wall remote from the crista, 3 (5%) on the mitral annulus, 3 (5%) at ostia of the pulmonary vein, and 2 (3%) in other left atrial sites.

Results: AT pts with SHD were older (61.8 ± 15.2 vs.41.7 ± 18.1 yrs, $p < 0.05$) than non SHD pts. Mean tachycardia cycle length, incidence of multiple foci, inducibility with programmed stimulation, and catecholamine requirement for sustained tachycardia did not differ between groups. In pts with SHD, AT was less likely terminated by adenosine (50% vs 83%, $p < 0.05$). However, AT in pts with SHD were less likely to arise from the left atrium or crista and more likely from other right atrial sites ($p < 0.05$).

Ablation Site	SHD	No SHD
Left atrial ATs	2 (8%)	6 (18%)
Right crista ATs	7 (28%)	18 (54%)
Right non-crista ATs	16 (64%)	9 (27%)

Conclusion: ATs in pts with SHD are more likely to originate from right atrial sites remote from the crista terminalis and are less likely to be adenosine sensitive than ATs in pts with structurally normal hearts.

1123-164 The Role of Ventriculoatrial Interval and Preexcitation Index in the Diagnosis of Supraventricular Tachycardia Mechanism

T.J. Lobo, P.A. Friedman, R.F. Rea, S.C. Hammill, W.-K. Shen, M.S. Stanton, T.M. Munger, M.J. Osborn, D.L. Packer. *Mayo Clinic, Rochester, MN, USA*

Background: Small studies in the pre-ablation era showed that the preexcitation index (PI; supraventricular tachycardia [SVT] cycle length minus the longest VPD coupling interval during SVT causing atrial preexcitation) and the ventriculoatrial conduction time (VA time) can suggest SVT mechanism as well as accessory pathway (AP) location. We reassessed these criteria in a large group of patients whose SVT circuits were defined by catheter ablation.

Methods: We evaluated the relation among SVT mechanism, AP location (when present), and PI and VA time in 208 patients undergoing radiofrequency (RF) catheter ablation with a single SVT mechanism and postablation QRS ≤110. Typical atrioventricular nodal reentry (AVNRT) was present in 125, and orthodromic reciprocating tachycardia (ORT) in 143 (98 left, 7 right and 38 septal AP's).

Results: VA time and PI were significantly different among groups (ANOVA) as were all pairwise comparisons (Fisher's PLSD) except septal vs. right AP's for both VA time and PI.

(msec)	AVNRT	Left AP	Right AP	Septal AP
PI	133 ± 39	88 ± 27	56 ± 15	50 ± 22
VA time	31 ± 22	87 ± 22	119 ± 41	115 ± 64

A criterion of PI >65 and VA time <65 identified 94.3% of AVNRT and excluded 100% of right and septal AP's and 96% of left AP's.

Conclusion: VA time and PI are useful in differentiating SVT mechanism and AP location. A combined criterion may be more useful in separating AVNRT from ORT.

1123-165 Effect of a Change in Rate on Atrial Repolarization

R. Goyal, Z.A. Syed, P.S. Mukhopadhyay, B.C. Tran, J. Souza, A. Zivin, F. Pelosi, M. Flomming, B.P. Knight, K.C. Man, S.A. Strickberger, F. Morady. *University of Michigan, Ann Arbor, MI, USA*

When pacing cycle length (CL) decreases, the resultant decrease in ventricular refractory period may take several mins to become fully manifest. No previous study has evaluated the response of atrial repolarization to sudden changes in rate. This study evaluated the effect of changing the rate of right atrial (RA) pacing on the maximum change and time course of atrial repolarization using monophasic action potential (MAP) recordings. 8 patients (age 38 ± 11 yrs) with structurally normal hearts undergoing a clinically-indicated electrophysiologic procedure were enrolled. A catheter was placed in the high RA and a MAP catheter (model#1675P, EP Technologies) at the interatrial septum. Action potential duration-90% (APD_{90}) was calculated from the digitized MAP recordings. All patients were given autonomic blockade (propranolol 0.2 mg/kg & atropine 0.02 mg/kg). The RA was paced in 5 min sequences at CL's of 500, 400, 500 and 300 msecs. There was a significant decrease in APD_{90} when pacing was decreased from 500 to 400 msec (235 ± 24 vs. 207 ± 17 msec, $p < 0.05$, ANOVA) and 32 secs was required for a stable value to be achieved. In contrast when pacing was decreased from 500 to 300 msec the duration for a stable APD_{90} value was only 18 sec (234 ± 28 vs. 153 ± 33 msec, $p < 0.05$). In conclusion, similar to ventricular tissue, the magnitude of APD shortening is dependent on the magnitude of change in the PCL. However, unlike ventricular tissue, the time required for stabilization is shorter when the change in pacing rate was 200 vs. 100 msec. These changes are independent of autonomic tone. This electrophysiologic difference between atrial and ventricular tissue may be due to the higher concentrations of I_{to} channels in atrial tissue.

1124 Ventricular Tachycardia: Cardiomyopathy; Mapping

Tuesday, March 31, 1998, Noon–2:00 p.m
Georgia World Congress Center, West Exhibit Hall Level
Presentation Hour: 1:00 p.m.–2:00 p.m.

1124-167 A New Index of Repolarization Lability Detects Mutation-specific Differences in Hypertrophic Cardiomyopathy Patients

W.L. Atiga, L. Fananapazir[1], D. McAreavey[1], H. Calkins, R.D. Berger. *Johns Hopkins Medical Institutions, Baltimore, MD, USA; [1]NIH, Bethesda, MD, USA*

Background: Certain β-myosin heavy chain mutations that cause familial hypertrophic cardiomyopathy (HCM) are associated with an increased risk of malignant ventricular arrhythmias. We tested whether patients with these mutations exhibit labile ventricular repolarization based on beat-to-beat QT interval variability analysis.

Methods: We measured the QT variability index (QTVI) and heart rate-QT interval coherence (COH) in 36 HCM patients and 26 age-and sex-matched controls. Genetic abnormalities in HCM patients included 7 different mutations. Nine patients had the malignant 403Arg→Gln mutation and 8 had the benign 908Leu→Val mutation. Five-minute epochs from Holter monitor recordings were digitized for QTVI and COH analysis.

Results: The QTVI was higher in HCM patients than in controls (−1.24 ± 0.42 vs −1.58 ± 0.38, $p < 0.01$). Also, in 403Arg→Gln patients, the QTVI was higher than in controls (−0.997 ± 0.49 vs −1.46 ± 0.43, $P < 0.05$), while in 908Leu→Val patients, the QTVI did not differ from controls (−1.44 + 0.44 vs −1.59 ± 0.45, $p = 0.5$). COH was lower in all HCM patients and those with the 403Arg→Gln mutation than in controls ($p < 0.001$ and $p < 0.05$, respectively).

Conclusions: Patients with familial HCM, especially those with a mutation associated with high arrhythmic risk, exhibit labile ventricular repolarization quantified by QT variability analysis.

TUESDAY POSTER